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SYPHILIS OF THE LUNG:

WITH A REPORT OF THE ANATOMICAL FINDINGS IN A CASE.

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Syphilis of the lung is either congenital or acquired. In this paper only the latter form will be considered.

Whether we agree with the majority who hold that syphilis of the lung is rare in the adult, or with the few who consider it more common, we all must accept the opinion of the best authorities—that the condition does exist. Now, when it is taken into consideration that syphilis is curable and that the advanced cases of pulmonary tuberculosis, with which it is likely to be confounded, are, as a rule, incurable, one should be all the more careful to make no mistakes.

That syphilis of the lung is a very rare condition is almost universally conceded by men of large experience. Osler states that in twenty-five years he has not seen more than half a dozen specimens in which there was no question as to the nature of the trouble.[1] In 1898 the London hospitals and Royal College of Surgeons contained only ten specimens which seemed to be unquestionable examples of this disease.[2] Among 13,000 specimens in the Army Medical Museum at Washington there is not one of this disease.

There are others, however, who hold a contrary opinion. Porter considers it common in adults. Satterthwaite[3] thinks that lung syphilis is greatly underestimated by the general practitioner, and "almost unknown to many syphilographers." Pankritius, Schnitzler, and Grandidier have also found it by no means unusual.

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[1] Practice of Medicine.

[2] Fowler and Godlee. The Diseases of the Lungs.

[3] Boston Medical and Surgical Journal, 1891, vol. cxxiv.

Stengel[1] suggests that this difference of opinion depends largely upon the point of view of the observer—whether as a clinician or as a pathologist, the former believing that this condition is comparatively common, the latter that it is very unusual. It must be acknowledged that the pathologist has by far the best of it when it comes to producing convincing evidence. There is no disease, however, in which it is more necessary to carefully weigh the pathological findings with the clinical history before giving an opinion.

Without questioning their rarity, it may be well to mention one or two possible factors which have tended to reduce the number of cases reported. In the first place, it is most difficult to differentiate between pulmonary tuberculosis and pulmonary syphilis. This is well recognized, not only at the bedside, but in the pathological laboratory. In fact, the only absolutely certain point of difference in many cases is the presence of the tubercle bacillus, and even this unquestionable evidence of tuberculosis does not preclude the possibility of associated syphilis. Pulmonary tuberculosis is so common and syphilis of the lung thought to be so unusual that it seems probable that some of these cases, because of their very rarity, may slip by with a diagnosis of tuberculosis. Many cases of supposed phthisis give such a typical history and show such characteristic signs of the disease that it does not seem necessary to examine the sputum, or, even if the bacilli are not found after a few examinations, no change in the diagnosis seems called for. Satterthwaite[2] gives a very interesting example of such a case which fell into his hands and was subsequently cured by appropriate treatment.

The same line of argument is used in the post-mortem room. The condition is so much more likely to be tuberculous than luetic that it is almost always placed in that class unless pronounced syphilitic lesions of other organs are discovered.

TERTIARY LESIONS. While secondary lesions of the trachea have been occasionally observed,[3] there are, so far as I know, no records of such lesions in the bronchi. From the similarity of structure and function, however, there seems to be no reason why similar secondaries (mucous patches) might not be found, should an opportunity for their observation offer. The results of antisyphilitic treatment upon bronchitis occurring during secondary manifestations also serves to strengthen the theory of possible secondary bronchial involvement.

Tertiary lesions of the trachea and bronchi, consisting of gummatous infiltration of the submucous tissue, with consequent fibrous thickening and stenosis, are not unusual, and the lesions of the pulmonary parenchyma are solely of the tertiary type.

THE PERIOD INTERVENING BETWEEN THE PRIMARY INFECTION AND THE APPEARANCE OF PULMONARY LESIONS. Undoubted syphilitic disease in the lungs has been recorded as early as one and as late as twenty years after the initial sore. The most usual time is, however, from the fifth to the tenth year after the infection. Frank[1] and Schirren[2] have reported cases only a few months after infection. My own case showed comparatively recent lesions twenty-one years after infection.

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[1] University of Pennsylvania Medical Bulletin, 1903-1904.

[2] Loc. cit.

[3] Morrell MacKenzie. Diseases of the Throat and Nose, London, 1880, vol. i. p. 510.

THE LESIONS OF SYPHILIS OF THE LUNG. 1. Gumma. 2. Pneumonia. 3. Fibrosis.

1. Gummata are either single or multiple, round, elliptical, or irregular in shape, and vary in size from a hemp-seed to a goose-egg, and in consistency from a soft colloid to a firm fibroid or cheesy tumor.

They may be found in any part of either lung, but are more usual within the substance and in the vicinity of the root, though they may be at the apex or upon the surface (my own case). Many observers claim that the right middle lobe is the favorite location. Grandidier[3] reported 30 cases in which the right middle lobe was the seat in 27. Hirsch[4] quotes Pankritius' statistics of 105 cases, in which 76 were right-sided, 18 left-sided, and 11 bilateral.

A gumma may present any of the following appearances, depending somewhat upon its age: It may be a gelatinous mass or a nodule of a grayish-white or yellow or reddish color, usually surrounded by a more or less distinct capsule, formed through inflammatory changes, set up in the surrounding lung tissue. On section there is a tendency for the centre to bulge somewhat, which is considered to be of some value as a distinction from tubercle, there being apparently more elasticity in the periphery of the gumma.

Microscopically they are found, like gummata of other organs, to consist of round cells, large spindle-shaped or irregularly formed cells, epithelioid cells, giant cells (though these are not as numerous as in tubercles), fibroid interstitial tissue, and elastic tissue. Councilman[5] considers the essential process in the production of gumma in the lung to be a pneumonia with fibrinous exudation, accompanied by fibrous change in the alveolar walls, the whole subsequently undergoing caseation. There is a tendency to caseation and degeneration, beginning at the centre; young vessels spring from the periphery and grow toward the centre, and lead to absorption of the caseous nodule. If the absorption is complete a cicatrix is formed. It is also believed by some that the broken-down material may be discharged through a bronchus, and thus a cavity may result. Cavity formation is, however, very rare. That the anatomical diagnosis of pulmonary syphilis is very difficult and sometimes impossible has been, and still is, the opinion of the most experienced observers (Virchow, Ziegler,[6] and Ribbert[7]).

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[1] Wien. med. Presse, 1880, Bd. xxi. No. 38.

[2] Dermatol. Zeitsch., 1893-1894.

[3] Berlin. klin. Wochenshr., 1875, No. 15.

[4] Ueber die Syph. Erkrankung der Lunge, München, 1901.

[5] Johns Hopkins Hospital Bulletin, 1891, vol. ii., No. 11.

[6] Spez. path. Anat., 1898.

[7] Lehr. b. d. Path. Histol., 1896.

Dürck and Hektoen[1] believe that a distinction can usually be made by the presence of newly formed bloodvessels in gumma. Sometimes, even in quite necrotic areas, vessels may be seen. Tubercle, on the other hand, as is well known, never shows new blood cells, and those old ones which may have been caught in the growth soon become occluded by pressure or thrombosis.

It is impossible to distinguish between tubercle and gumma in some instances, except by the finding of the tubercle bacillus. In a general way it may be said that in gumma fatty degeneration plays a very important part, while in tubercle the hyaline form is the more usual, and that giant cells are much rarer in gumma than in tubercle.

The pulmonary tissue immediately surrounding a gumma is frequently found to be consolidated, and from this infiltrated area fibrous bands may be seen extending to other parts of the lung.

2. Syphilitic Pneumonia of the Adult. It is pretty generally conceded that there is an infiltration of the lungs resulting from syphilitic infection, which alone, or in connection with nodules, may extend over a pulmonary lobe.

On section these infiltrated areas are of a dark slate or gray or grayish-red color, not granular.

Microscopically, as described by Aufrecht,[2] the alveoli were seen to be filled with cellular material, a few containing swollen alveolar epithelia exclusively; slightly more numerous were alveoli in which a small number of white blood cells were embedded in fibrinous masses, while the alveolar capillaries were distended with blood. But most of the alveoli appeared to be filled with small round cells interspersed with red blood corpuscles. A much larger quantity of the latter was present in the interalveolar and interlobular tissue. According to the same author, however, the only justification for considering this pneumonic inflammation as entitled to a special classification would be a considerable increase in the thickness in the walls of the smaller vessels.

3. Fibroid Induration. The proliferation of the connective-tissue elements may take the form of a thickening extending from the hilus around the bronchi and vessels, or of isolated masses of fibroid tissue in various parts of the lung, or of diffuse changes occupying the whole or the greater part of one lung.

This primary proliferation of the connective tissue in lung syphilis is considered by Pankritius[3] as being characteristic of lues, for, while in other diseases of the lungs which have a hyperplasia of the connective tissue, it is secondary to some primary condition, such as inhalation of dust, protracted croupous pneumonia, certain forms of tuberculosis, carcinoma, etc.

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[1] Hektoen's Trans. of Dürck's General Path. Hist.

[2] Nothnagel's Cyclopedia of Practical Medicine.

[3] Hirsch, Jos. Inaugural Dissertation, München, 1901.

Microscopically this change is characterized by hyperplastic cellular proliferation of the interstitial, interalveolar, peribronchial, and perivascular tissues. The vessels show a thickening of the intima and the adventitia. The favorite seat of this form of the disease, as in the case of gummata, is said to be in the right middle lobe.

Virchow has pointed out that diffuse lung syphilis cannot be distinguished from the chronic pneumonia of stone-cutters and grinders.

SYPHILITIC PHTHISIS. *A Progressive Destructive Disease Due to Syphilis.* The possibility of such an entity as a result of syphilitic infection has not yet been satisfactorily proven. Osler says: "Personally, I have no knowledge of such an affection, either clinically or anatomically, and the cases which I have seen demonstrated do not seem to me to have characteristic distinction enough to separate them from ordinary tuberculous phthisis."

Until our knowledge of syphilis of the lung is much more exact than it is at present, we must agree with Fowler that before cases are put in this category they should fulfil the following conditions:

1. The cases must be complete—that is, the symptoms observed during life must be considered in connection with the lesions found on post-mortem examination.
2. The evidence of syphilitic infection must be undoubted.
3. Repeated examinations of the sputum must have been made, and the tubercle bacilli have been invariably absent, and the absence of tubercle from the lungs (as the cause of lesions) must be proved by post-mortem examination.
4. Syphilitic lesions about the nature of which there can be no doubt must be found in other organs. He gives several cases which seem to fulfil these conditions.

Of course, it is quite possible for a lobe or entire lung to be excavated as a result of more or less complete bronchial obstruction by a gumma, just as is occasionally seen in cases of aneurysm, but this is not due to the effect of the syphilitic virus upon the parenchyma.

It would hardly seem justifiable to classify separately certain changes observed in the bronchial glands and lymphatics in a case of syphilis of the lungs and other organs reported by Weber.[1]

SYMPTOMS. Cough is usually the first symptom to attract attention, but has nothing characteristic. It depends more upon the laryngeal, tracheal, and bronchial lesions than upon those of the lung proper; at least, until these have become serious.

There is in many of these cases an associated ulceration of the larynx, trachea, or bronchi, which causes more or less constant irritation, resulting in cough.

Sputum. At first the sputum is that of an ordinary bronchitis, but later may become fetid from the bronchiectatic cavities formed by the syphilitic changes in the lung. No tubercle bacilli are present in uncomplicated cases. There may, however, be elastic tissue depending upon the breaking down of the pulmonary parenchyma.

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[1] Pathological Society Transactions, vol. xvii. p 152.

Hœmoptysis. This symptom, though not common, does occur, and is one of the most misleading, because it is so very usually a sign of tuberculosis (probably 5 out of 6 cases of pulmonary hemorrhage are due to tuberculosis). The amount of blood lost is not, as a rule, large, though a patient of Lancaux expectorated more than a litre in twenty-four hours.

Dyspnœa. This seems to be very usual. Of course, the frequently associated stenosis of the trachea and bronchi (pressure by gummata on cicatricial contraction) accounts for this symptom in many cases, but it sometimes seems out of proportion to the anatomical lesions. Mauriac explains its origin in these cases as due to flattening of the alveoli and degeneration of the alveolar epithelium. The shortness of breath is often paroxysmal and resembles at times bronchial asthma.

Pain. This is not considered an important symptom by the majority of observers.

GENERAL SYMPTOMS. *Fever.* The temperature is of no value for diagnostic purposes, as it may be high or fever may be absent entirely. When present it may be of the hectic type, as in tuberculosis.

Night-sweats have been noted, but are not so common as in tuberculosis.

Emaciation, though not as frequent as in phthisis, may, in advanced cases, be most pronounced.

PHYSICAL SIGNS. There is, unfortunately, nothing distinctive. The signs produced by consolidation, whether from gummata or infiltration of syphilitic origin, differ in no way from those of tubercles or tuberculous infiltration.

Bronchiectatic cavities or excavations in the parenchyma produce the same signs, no matter from what cause.

DIAGNOSIS. Some writers, especially the German, lay great stress upon the much greater frequency of syphilitic lesions in the middle lobe of the right lung. Grandidier, having observed this apparent preference in 27 out of 30 cases, considers it of the greatest importance in making a diagnosis. Schnitzler and Pankritius have made the same observation. On the other hand, Fowler[1] says: "A careful examination of undoubted specimens of pulmonary syphilis does not bear out the statement that lesions are generally limited to the middle part of the lung; they are so often found elsewhere that little importance attaches to the exact size in deciding the question of diagnosis."

A physical examination of the chest, taken with the symptoms,

[1] Loc. cit.

usually suggests tuberculosis, and this is the diagnosis made in the vast majority of cases. The absence of tubercle bacilli, after repeated examinations of the sputum, first suggests the possibility of syphilis. If a history of lues has not already been elicited, careful questioning may now bring it out, and a suspicion of the real cause of the trouble be aroused. The seat of the primary lesion may be found; the history of a cutaneous eruption, sore throat, or falling of the hair be given. Ulceration of the pharynx or larynx may be noted or probably luetic lesions of the bones, such as the sinking in of the bridge of the nose. Advanced arteriosclerosis in a young patient is also suggested. The signs and symptoms of stenosis of the trachea or main bronchi, where an aneurysm or growth can be excluded, may also strongly indicate syphilis as the probable cause.

PROGNOSIS. In early cases the prognosis is favorable. Unfortunately, a correct diagnosis is usually reached so late in these cases that the disease is many times too far advanced to admit of a cure. Whether antisyphilitic treatment will do much toward the removal of a gumma which is already in the advanced fibroid or cheesy state seems doubtful. The effect of treatment upon those chronic fibroid changes about the bronchi in the alveolar walls seems problematic in the extreme. The process may, however, be arrested. Bronchiectatic cavities are always serious, but when the obstruction is due to gummatous thickening, and not to fibroid contraction following submucous gummatous ulceration, it is probable that the condition may be cured; provided, of course, that it has not existed too long a time. If real cavity formation has occurred, the prognosis is unquestionably bad.

TREATMENT. Antisyphilitic treatment should be commenced without a moment's delay. The general trend of the present day is toward the use of mercury, as well as the iodides, in tertiary lesions, no matter where situated.

The administration of blue ointment by inunction and of potassium iodide by the mouth seems the best and most rapid way of combating this very serious disorder.

A great deal has been said about the occurrence of syphilis of the lung in conjunction with pulmonary tuberculosis. It may be said that it would seem the wisest plan, when such a condition is suspected, to institute the antisyphilitic treatment, with the hope of removing that infection at least. Cases have been reported which show that an antisyphilitic treatment may be carried out and the symptoms, such as roseola and ulceration of the pharynx, disappear without any ultimate bad results, so far as the tuberculous conditions are concerned.

The following report of a case of syphilitic disease of the lungs is of interest chiefly from a pathological point of view, as there were no symptoms which suggested the possible existence of the post-mortem findings:

William D., white, aged forty-four years; American; farmer. One uncle died of tuberculosis; otherwise the family history was good. He never drank whiskey and seldom beer; used tobacco moderately. At twenty-three years developed a sore upon the penis after exposure, the scar of which still remained. He received internal treatment at once and no secondaries developed. At thirty-nine years, sixteen years after the primary sore, he had a stroke, which paralyzed his right arm and the right side of his face. In about a year the paralysis was completely recovered from. Nine months ago he suffered from a swelling of the abdomen (probably ascites), for which he was treated and recovered.

For the past year he had been obliged to rise each night to urinate. For more than a month he had noticed that his feet were swollen, especially in the morning. Some months ago he ran a pitchfork into his ankle, making a small, punctured wound, which had never healed.

The present attack began, about two weeks before admission to the Garfield Hospital, with a cutting pain in the chest; a week later pain began in the abdomen. The feet and abdomen then became noticeably swollen, and there was dyspnœa on exertion. There was a cough with mucopurulent expectoration, which contained no tubercle bacilli.

The skin was pale, the eyelids puffy; there was general œdema. No thickening of the radials could be detected. The pulse tension was slightly plus. The heart was not enlarged; the second aortic sound accentuated; there were no murmurs. The respiratory sounds were harsh. The abdomen contained a fair amount of fluid. The thoracic and abdominal veins were distended. No enlargement of the spleen could be detected. The liver dulness seemed to be diminished, probably due to the floating up of the intestines by the ascitic fluid. The edge of the liver could not be felt.

Upon the sternum, between the junctions of the second and third ribs, was an irregularly shaped, flat swelling, which was not painful, giving to the examining finger a sense of hard elasticity. It was said to have followed a kick by a horse, and, as it was not recognized as a gumma, but little attention was given it.

The urine was scanty, averaging about 600 c.c. in the twenty-four hours. It was turbid, acid, specific gravity 1026, albumin two-thirds by bulk, no sugar; hyaline, fatty, granular, hyeloepithelial casts; a few leukocytes and blood cells. Ten and two-tenths grams of urea were excreted in twenty-four hours.

The urinary findings continued about the same through the course of the illness.

The blood showed 73 per cent. of reds, 8700 leukocytes, and 65 per cent. of hæmoglobin.

The œdema gradually became more pronounced, the ascites increased, and a left pleural effusion developed.

Treatment was of no avail, and the man died.

The clinical diagnosis was chronic parenchymatous nephritis, possibly of syphilitic origin.

I was present at the autopsy, which was performed by Dr. Butterfield. The notes read as follows:

Projecting from the sternum, between the second and third ribs, is an irregularly round nodule of firm consistence, not movable beneath the skin, about 3 or 4 cm. in diameter and 1.5 cm. in height. A pale, depressed scar, 1.5 cm. in diameter, on under surface of glans penis.

Inguinal glands enlarged and easily palpable. On the dorso-internal aspect of the left foot are two reddish areas covered with yellow crusts (scars from wounds mentioned in history).

Hypostasis in dependent portions. No evidence of cutaneous œdema. Parietal peritoneum thickened and opaque. Section of sternal nodule firm, yellow, surface smooth. It is firmly attached to sternum and adjacent ribs.

Pleural membranes thickened and opaque. Right cavity contains normal amount of fluid. Left cavity contains 45 to 60 c.c. of reddish serum.

Left Lung. Visceral pleura adherent at the apex anteriorly and posteriorly; also adherent between the lobes. On the anterior aspect of the upper lobe is a yellow nodule, 2.5 by 1.5 cm., adherent to the overlying costal cartilage. On section it appears to be encapsulated, extends down into the lung substance, is firm and is traversed by lines of pigment. The lung is crepitant throughout, slightly pale, and emphysematous along the anterior border and purplish-red at the base. Pigment deposits in septa; on section frothy serum exudes. Bronchi contain a mucopurulent material (cover-slip preparations show no tubercle bacilli). Weight, 505 grams.

Right Lung. Extensive adhesions at the apex, posterior portion of the upper lobe; also to diaphragmatic pleura. Pleura between the lobes adherent. On the inner aspect of the upper lobe is a yellow nodule, presenting the same general characteristics as that described in connection with the left lung. Lung crepitant throughout, pigmented pinkish. Bronchi contains mucopus. No tubercle bacilli demonstrable. Weight, 550 grams. No enlargement of the bronchial glands.

Pericardium. Parietal membrane thick, opaque. Cavity contains the normal amount of fluid. Heart apex is adherent at the base of the pericardial triangle. Epicardial membrane shows whitish opaque areas irregularly distributed over the heart. Myocardium thin, slightly flabby, and of a dark reddish-brown color. Aorta contains numerous atheromatous patches; on the aortic valve cusps are a few small vegetations. One leaflet of the mitral valve thickened and slightly contorted; the other slightly thickened. Other valves normal. Weight of heart, 241 grams.

Parietal peritoneum thick and opaque. Localized adhesions in some portions of visceral peritoneum. No excess of fluid. One large nodule in the omentum. No evidence of fat-necrosis.

Liver. Extensive adhesions to the diaphragm. Surface pale, marked by opaque, slightly depressed areas, a few mm. in diameter. Yellow encapsulated nodules in right lobe posteriorly. One extends into the liver substance for more than 3 cm. One very prominent nodule in the left lobe just at the fissure anteriorly. On section it is pale, firm, yellow, and showing a tendency to bulge in the centre of the nodule. Gall-bladder tense, with thick, greenish-black bile; not enlarged; no stones. Weight, 1668 grams.

Pancreas. Dense and enlarged; adherent to the spleen. Surface marked by numerous, whitish, opaque, miliary areas. Section firm; connective tissue between lobes increased and contains the same white opaque areas seen on surface of the viscus. Weight, 131 grams.

Kidneys. Right organ. Apex and adrenal body adherent to the liver; perirenal fat very abundant. Organ enlarged (12 x 6 x 4.3 cm.); surface smooth, pale, and mottled venæ stellatæ injected. Section showed no increased resistance on cutting. Cortex 9 mm. in width, pale, with yellowish areas. Pyramids congested, being of a dark purplish-red color.

Fat about the pelvis increased. Capsule strips readily. Weight, 293 grams.

Left Kidney. The same general characteristics as the right. Measurements, 13 x 6.5 x 4.5 cm. Weight, 294 grams.

Spleen enlarged, firm, but friable; surface smooth and capsule opaque. Section firm, deep red in color. Weight, 320 grams.

Stomach and intestines apparently normal. On the surface of the bladder there is a small nodule corresponding in consistence and color to those described in the liver, lungs, omentum, and upon the sternum.

MICROSCOPIC FINDINGS. The nodule upon the sternum showed the usual picture of a gumma.

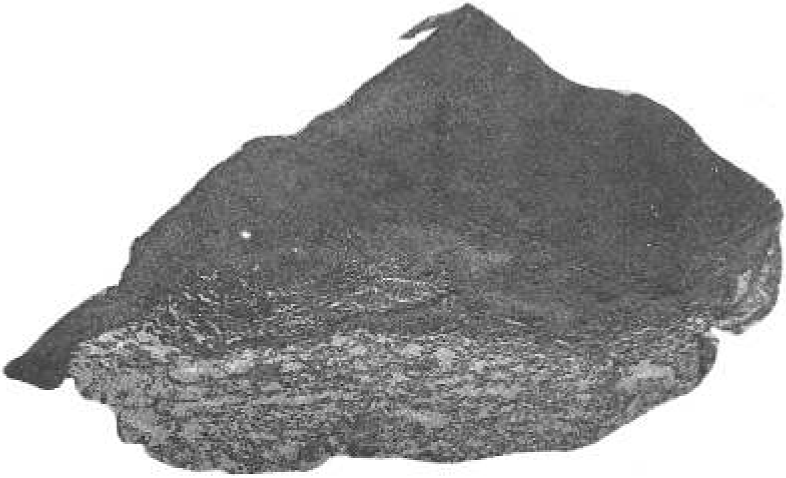
Sections of the liver showed marked cirrhosis, gummata, and amyloid degeneration. The kidneys were the seat of a diffuse nephritis and amyloid change. The pancreas showed pronounced cirrhosis of the interlobular variety and areas of fat-necrosis. No sections were cut from the nodule in the omentum, nor from that upon the bladder, but the macroscopic appearance of all the nodules was the same, and I have no doubt that they were all gummata.

The Lungs. The two nodules described as being situated upon the anterior and inner aspects of the upper lobes of the right and left lungs, respectively, were found on section to consist of dense fibrous tissue, with necrotic centres (Fig. 1).

There was no distinct capsule on that portion of the growth toward the lung tissue. The alveoli seemed to fade so gradually into the continuous fibrous growth that it was difficult to detect any distinct dividing line.

Numerous bloodvessels could be seen in the connective tissue (Fig. 2), and even a few in the necrotic area. This point is especially worthy of note, as it is considered by Dürck and Hektoen as characteristic of gumma in contradistinction to tubercle. The latter never show new bloodvessels. From the fibrous tissue of the nodule could be seen bands of connective tissue dipping down into the parenchyma of the lung, which in places showed a condition of catarrhal pneumonia (Fig. 3). On the outer aspect of the growth the fibrous tissue was continuous with the thickened pleura.

FIG. 1.

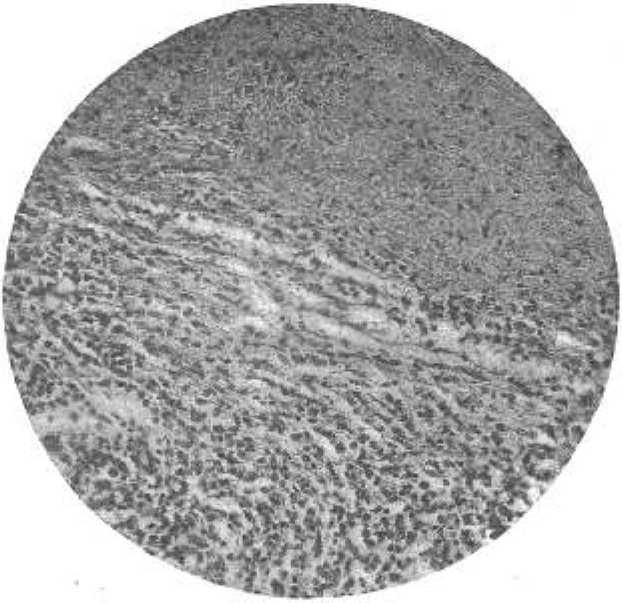


A section through part of a small gumma of the lung. × 10.

A number of sections were made from both lungs, which showed irregularly distributed areas of fibrosis and also small areas of pneumonia. These consolidated areas were too minute to be detected macroscopically, nor could they be felt. There was a thickening of the adventitious coats of the arteries, but none of the intima. As to whether or not there was any actual increase of fibrous tissue about the bronchi, I was unable to convince myself; it was certainly not universal.

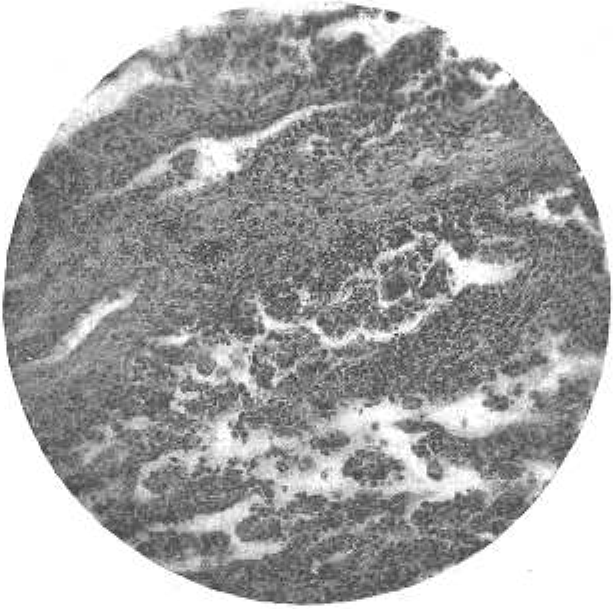
The belief that the nodules in the lungs were gummata is based on the following: 1. History of a chancre, the scar of which remained. 2. The history of a paralytic stroke at thirty-eight years. 3. The presence of undoubted gummata of the liver and on the sternum. 4. The absence of tubercle bacilli from the sputum before death and at post-mortem. 5. The absence of bacilli from the sections of the nodules. 6. Extensive amyloid change in the liver, spleen, and kid-

FIG. 2.



Showing part of the new-growth, with granulation tissue rich in capillary bloodvessels at the margin. × 200.

FIG. 3.



A, Field beyond the fibrous area, showing a condition of catarrhal pneumonia and increased connective tissue in the area adjacent to the new-growth. × 125.

neys. 7. The absence of any tuberculous involvement of the apices or any other part of either lung. 8. The presence of numerous bloodvessels in the new-growth.

Although the presence of considerable black pigment would suggest a possible cause for the fibrosis in the lungs, it was noted that the fibroid areas apparently bore no constant relationship to the patches of pigment, and, therefore, it seems fair to conclude that the scattered areas of fibrosis and of consolidation were also of syphilitic origin.

I wish to acknowledge my indebtedness to Dr. James Carroll for his aid and advice in interpreting the microscopic findings.

SOME ASPECTS OF THE THEORIES OF IMMUNITY.

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On infection by bacteria it is the bacterial toxins which cause disease, the bacteria as such merely representing inert foreign matter. There are two distinct classes of bacterial toxins:

1. *Extracellular toxins* secreted by the bacteria during growth. Such toxins dissolve out into the surrounding medium, causing disease and death by toxæmia. Examples, diphtheria and tetanus.

2. *Intracellular or endotoxins,* which can hardly be regarded as secretions, but rather as a part of the bacterial body substance, being only liberated on the death and dissolution of the bacteria.

In the latter case the bacteria on gaining entrance to the circulation or body fluids may be destroyed at once, the amount of toxin thus liberated being too small to affect the organism, or the bacteria may be able to multiply; progressive increase and destruction going on *pari passu* until the number being destroyed is so great that the intracellular or endotoxins are liberated in sufficient quantity to cause manifestations of disease. Radziewski[1] was the first to point out this principle in a clear and convincing manner. An infection by such bacteria may be circumscribed and express itself as a local inflammatory process or it may be generalized in the circulation, in which case it is called septicæmia or bacteræmia. It will be convenient here to speak only of bacteræmia. Examples: Typhoid fever, staphylococcus and streptococcus infections.

The processes of immunization against these two types of infection are entirely different:

1. In toxæmia antitoxins are formed which neutralize the toxin, but do not affect the bacteria.